



determined by the date of last menstruation. Body mass index (BMI) was calculated by using the formula of weight in kg/h (m<sup>2</sup>) (height and weight were measured by using the height rod attached to the balanced beam scale), values taken were <20 kg/m<sup>2</sup> (low), 20-25 kg/m<sup>2</sup> (normal), and >25 kg/m<sup>2</sup> (high). USG was performed in the Radiology Department of the same institute by the radiologist on duty on the same USG machine to study placental location and dimensions. Women were followed-up with USG (at 20<sup>th</sup> and 30<sup>th</sup> week) to diagnose fetal growth restriction (FGR) or other abnormalities and clinically for maternal/neonatal outcome in terms of disorders like hypertensive disorders, gestational diabetes mellitus (GDM), placental abruption, cesarean sections, FGR, and preterm births. The findings on each visit were recorded on a predesigned proforma. Overall, 915 women were enrolled in the study, with 70 dropouts, 28 first trimester, and 16 second trimester abortions (analyzed separately); 801 study subjects were followed for maternal neonatal outcome. Statistical analysis was done by Epi 6 software (6.0, developed by Centres for Disease Control and Prevention, Atlanta, Georgia, USA) using Chi-square test and Fischer exact test for determining the statistical significance of the observations. P values of <0.05 were considered as significant.

Placental location was recorded and, according to major area of attachment, it was labeled as anterior, posterior, or fundal, irrespective of its extension to lateral side in anterior, posterior, and anterior or posterior in cases of fundal placenta and the area was measured by the maximum longitudinal dimension taken as the diameter of the placenta and the thickest part of the placenta, wherever it was (usually near the cord insertion), as the height. The placental thickness was calculated by taking the average of the three best measurements through the probe, oriented to scan perpendicular to the placenta. The placenta was labeled thin if the thickness was less than the tenth percentile, as normal if it was between the tenth and the ninetieth percentile, and as thick if it was more than the ninetieth percentile.<sup>[7]</sup>

## RESULTS

Out of the 801 women, 200 (25%) had mainly anterior placenta, 123 (15.4%) posterior, in 322 (40.2%) major part was fundal, and, in the rest, placenta was in the lower part of the uterus, 129 (16.1%) Grade I, 9 (1.12%) Grade II, 6 (0.8%) Grade III, and 12 (1.5%) Grade IV placenta previa.<sup>[8,9]</sup>

Of the 78 women with BMI <20, 29 (37.2%) had anterior, 9 (11.5%) posterior, 28 (35.8%) fundal, and 12 (15.4%) had placenta covering the internal os; 14 (18%) had thick placenta, 16 (20.5%) thin, and 48 (61.5%) women had normal placenta; 29 (37.2%) had placental surface area <41 cm<sup>2</sup>, 36 (46.2%) had 41-55 cm<sup>2</sup>, and 13 (16.7%) had >55 cm<sup>2</sup>.

Of the 665 women with BMI between 20 to 25, 158 (23.7%) had anterior, 105 (15.8%) had posterior, 267 (40.2%) had fundal placenta, and 6 (0.9%) had placenta covering the os; 124 (18.6%) had thick placenta, 72 (10.8%) thin placenta, and 469 (70.5%) had normal placenta; 156 (23.5%) had placental surface area <41 cm<sup>2</sup>, 346 (52.0%) had 41-55 cm<sup>2</sup>, and 163 (24.5%) >55 cm<sup>2</sup>.

Of the 58 women with BMI >25, 13 (22.4%) had anterior, 9 (15.5%) posterior, 27 (46.6%) had fundal placenta, and 9 (15.5%) had low-lying placenta; 10 (17.2%) had thick placenta, 8 (13.8%) thin, and 40 (69.0%) had normal placenta [Table 1]; 12 (20.7%) had placental surface area <41 cm<sup>2</sup>, 32 (55.2%) 41-55 cm<sup>2</sup>, and 14 (24.1%) >55 cm<sup>2</sup> [Tables 1-3].

**Table 1: Age, BMI and placental thickness**

Age (yrs)	BMI	Placental Thickness					
		Thick		Thin		Normal	
		No.	%	No.	%	No.	%
≤19 (37)	<20	5	13.5	4	10.8	16	43.2
	20-25	4	10.8	1	2.7	1	2.7
	>25	4	10.8	2	5.4	0	0
20-29 (736)	<20	5	0.7	5	0.79	22	3.0
	20-25	116	15.8	66	9.0	470	63.9
	>25	6	0.81	6	0.8	40	4.1
>30 (28)	<20	4	14.3	7	25	10	35.7
	20-25	4	14.3	0	0	3	10.7
	>25	0	0	0	0	0	0
801		148	18.5	91	11.4	562	70.2

BMI – Body mass index

**Table 2: Age, BMI, and placenta location**

Age (yrs)	BMI	Placenta location									
		Anterior		Posterior		Fundal		Low lying (I and II)		Covering os (III and IV)	
		No.	%	No.	%	No.	%	No.	%	No.	%
≤19 (37)	<20	6	16	3	8.1	0	0	2	5.4	1	2.7
	20-25	8	21.6	5	13.5	0	0	4	10.8	5	13.5
	>25	0	0	1	2.7	1	2.7	1	2.7	0	0
20-29 (736)	<20	24	3.4	19	2.6	168	22.8	20	2.7	3	0.4
	20-25	124	16.8	81	11.0	124	16.8	107	14.5	2	0.3
	>25	18	2.4	10	1.4	25	3.4	4	0.5	7	1.0
>30 (28)	<20	3	10.7	3	10.7	3	10.7	0	0	0	0
	20-25	8	28.6	1	3.6	0	0	0	0	0	0
	>25	9	32.1	0	0	1	3.6	0	0	0	0
801		200	25.0	123	15.4	322	40.2	138	17.2	18	2.2

BMI – Body mass index

**Table 3: Age, BMI and placental surface area**

Age (yrs)	BMI	Placental surface area					
		41 cm <sup>2</sup>		41-55 cm <sup>2</sup>		>55 cm <sup>2</sup>	
		No.	%	No.	%	No.	%
<19	<20	4	10.8	4	10.8	4	10.8
	20-25	4	10.8	4	10.8	4	10.8
	>25	6	16.2	7	18.9	0	0
20-29 (736)	<20	80	10.9	108	14.7	97	12.6
	20-25	70	9.1	141	18.3	40	5.2
	>25	23	3.0	134	17.4	43	5.6
>30 (28)	<20	3	10.7	5	17.9	3	10.7
	20-25	5	17.9	3	10.7	2	7.1
	>25	0	0	0	0	0	0
801		195	24.3	406	50.7	200	25.0

BMI – Body mass index

With anterior placenta, placental abruption occurred in 5 (2.5%), hypertensive disorders in 5 (2.5%), none of the babies of these women had FGR, 10 (5%) women had spontaneous preterm labor, and the rest 180 (90%) women had no abnormalities during pregnancy and labor. Of 322 women with fundal placenta, 66 (20.5%) had hypertensive disorders and 28 (42.4%) had FGR, 22 (7%) had placental abruption, 34 (10.6%) had preterm births, and 200 (62.1%) had no disorders. With posterior placenta, hypertensive disorders occurred in 12 (9.5%) (4 of these 12 had FGR also), placental abruption occurred in 4 (3.3%), 18 (14.6%) women had spontaneous preterm labor, and 85 (69.1%) had no abnormalities during pregnancy or birth [Figure 1].

Of 195 with area <41 cm<sup>2</sup>, 37 (19.0%) had hypertensive disorder, and 22 (11.3%) had placental abruption; with area 41-55 cm<sup>2</sup>, 30 (7.2%) had hypertensive disorders and 21 (5.0%) had placental abruption; with area >55 cm<sup>2</sup>, 13 (6.8%) had hypertensive disorders and 7 (3.7%) had placental abruption [Figure 2].

Of the 148 women with thick placenta, 58 (39.2%) had hypertensive disorders [22 (38%) had FGR also], 17 (12.2%) had placental abruption, and 11 (7.4%) had GDM. Of the 96 women with thin placenta, 9 (9.4%) had hypertensive disorders [of whom 2 (22.2%) had FGR also], 16 (16.7%) had placental abruption, and 9 (9.4%) had GDM [Figure 3].

Of 801 women, 91 (11.46%) had preterm births, of 200 women with anterior placenta, 0.5% (1/200), with posterior placenta, 14.6% (18/123) and with fundal placenta, 10.6% (34/322) had preterm births; there was no significant difference among the three groups ( $P < 0.001$ ).

With placental surface area <41 cm<sup>2</sup> (195), 56 (28.7%), with area between 41-55 cm<sup>2</sup> (416), 59 (14.2%) and with area >55 cm<sup>2</sup> (190), 30 (15.8%) had cesarean births. CS rate was highest in women with <41 cm<sup>2</sup> placental surface area ( $P < 0.001$ ). Comparing the CS rate for fetal distress in cases of thin (27%) and thick placenta (36.1%), the difference was not statistically significant ( $P = 0.16$ ), but no woman with normal placenta had CS for fetal distress, and the difference was highly significant ( $P < 0.001$ ).

## DISCUSSION

The birth of a healthy infant depends upon a coordinated series of events in the development of placenta and the fetus. Detailed analysis of gross placental structure can provide biologically relevant information regarding placental growth, development, and their potential consequences.<sup>[10]</sup> Over the years, USG has evolved as a safe noninvasive imaging technique for evaluation of fetal placental unit to detect and predict abnormalities, and it is

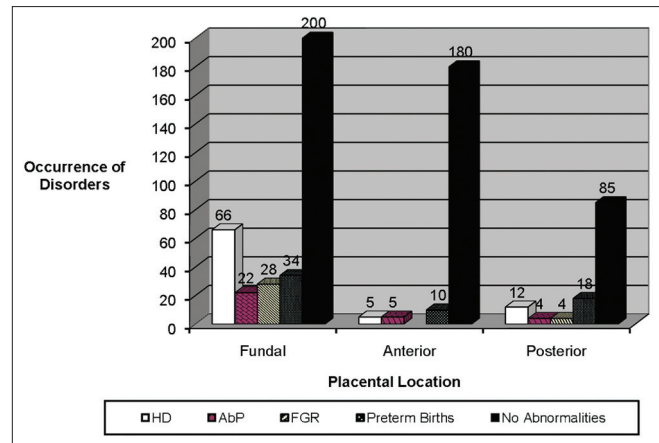


Figure 1: Placental location and disorders

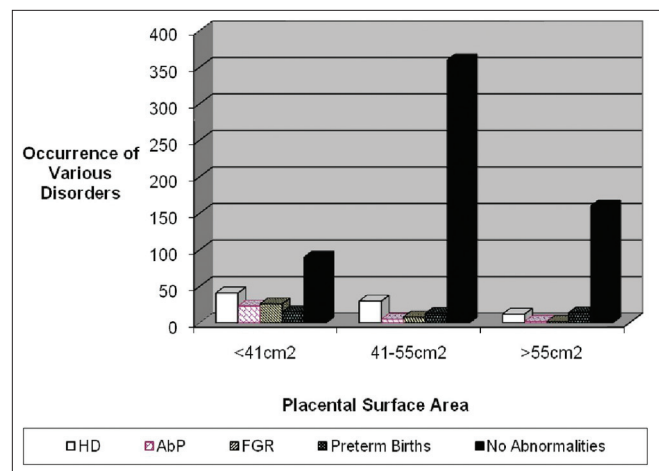


Figure 2: Placental surface area and disorders

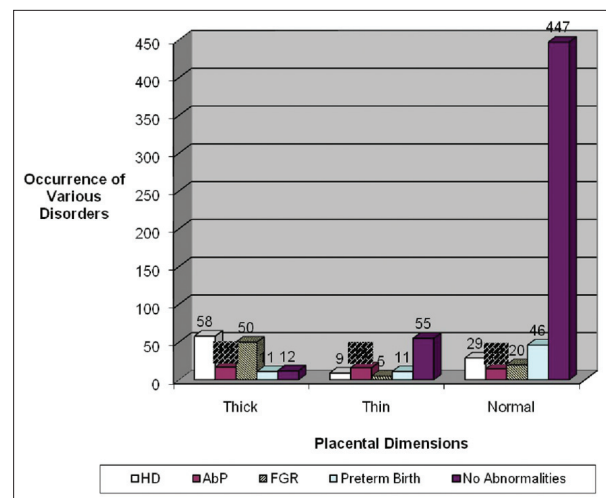


Figure 3: Placental dimensions and disorders

being used for fetal well-being by many researchers. Kinare et al.,<sup>[11]</sup> reported that, in Indian mothers, mid-pregnancy placental volume is significantly associated with pre-pregnancy maternal weight, and it is an independent predictor of birth weight.

Thame *et al.*,<sup>[12]</sup> provided evidence that both placental volume and the rate of placental growth may influence fetal size. Researchers have also reported that these effects are evident in the first half of pregnancy, and appear to be mediated through maternal weight and weight gain; however, studies about the placental location, dimensions in early pregnancy, and the maternal neonatal outcome are lacking. In the present study, placental location and dimensions at early gestation were studied to know their correlation with pregnancy outcome. Placental location has been implicated in preterm birth, in fetal malposition, in malpresentation, and in the development of pre-eclampsia.<sup>[13,14]</sup>

In the present study of total 801 women, 78 with BMI <20, 30 (38.46%) of them had anterior placenta, 10 (12.82%) had posterior placenta, and 38 (48.71%) had fundal placenta. With BMI 20-25 (389), 29 (7.45%) had anterior placenta, 60 (15.42%) had posterior placenta, and 156 (40.10%) had fundal placenta; with BMI >25 (334), 140 (41.91%) had anterior placenta, 58 (17.36%) had posterior placenta, and 136 (40.71%) had fundal placenta. It was revealed that, with anterior placenta, only 2.5% had hypertensive disorders and none of the women with anterior placenta, who later developed hypertensive disorders, had FGR as compared to women with fundal placenta in whom, 66 (20.5%) women had hypertensive disorders and 28 of them had FGR also. Five women (2.5%) with anterior placenta in the first trimester had placental abruption, 22 (7%) with fundal placenta, and 4 (3.3%) with posterior placenta ( $P=0.326$ , not significant). Depending on major location, anterior, posterior, or fundal part of placenta was labeled as anterior, posterior, and fundal; however, some had part of placenta antero-lateral, postero-lateral, fundo-posterior, or fundo-anterior; this was not analyzed separately. It has also been reported by other researchers that fundal or cornual placenta are risk factor for utero placental apoplexy, causing placental abruption, which may lead to poor maternal-neonatal outcome.<sup>[15]</sup> In the present study, it was revealed that fetal distress occurred more often in women with posterior and fundal placenta as compared to anterior placenta ( $P<0.001$ ), and, overall, there were more problems in cases of fundal placenta, followed by posterior as compared to anterior placenta. Kalanithi *et al.*, have reported that pregnancy complicated by IUGR are significantly more likely than non-IUGR pregnancies to have a lateral placenta as compared with an anterior or posterior placenta at 16-20-weeks gestation.<sup>[16]</sup>

Earlier, Khan *et al.*,<sup>[17]</sup> reported that overall 8% cases of the low-lying placenta had growth retarded babies as compared to 6% of the normal ones. Corneau *et al.*,<sup>[18]</sup> found no difference in the gestation of babies between low lying and normally sited placenta. Joseph *et al.*,<sup>[19]</sup> reported a statistically significant low incidence of hypertensive disorders in placenta similar to earlier reports. In the

present study, only one (8.3%) out of 12 women had placenta covering the os completely at term, had FGR and hypertensive disorders. There was no association with low-lying placenta. Whether placental migration occurs is debatable, and there are studies that reveal that migration does not occur in any patient with central placenta previa.<sup>[20]</sup> In the present study, migration issue of low-lying placenta was not studied in depth. However, in the final analysis, it was revealed that, for the major degree of placenta previa, there was no change in the location.

Hypertensive disorders occurred in 9.4% women with thin placenta as compared to 2.8% with normal placenta ( $P<0.001$ ). Hypertensive disorders and vascular disease can have linkage to placental structure. It has been reported that thin placenta is often a marker for small for date fetus or a sign of growth restriction, intrauterine infection, and preconception diabetes mellitus.<sup>[21]</sup> In the past, Hoogland *et al.*,<sup>[11]</sup> revealed a “warning limit” of placental area at mid-pregnancy. If placental area was equal/smaller than with the limit of 187 cm<sup>2</sup>, six of nine patients (67%) compared to four of 41 subjects with larger placentas ( $P<0.01$ ) had delivered a small-for-gestational age baby. Recent study by Schwartz *et al.*,<sup>[22]</sup> revealed that two-dimensional placental measurements taken in mid-gestation are significantly correlated with the incidence of small for gestational age (SGA). Elchalal *et al.*,<sup>[23]</sup> reported that perinatal mortality is significantly higher, (6.8%) in sonographically revealed thick placenta than in normal (0.66%). These findings are in accordance with earlier study by Williams *et al.*,<sup>[24]</sup> however, in William’s study, the placental thickness was measured in the second trimester. In a study by Luigi *et al.*,<sup>[25]</sup> where jelly-like thick placenta was diagnosed at early gestation and women were followed for the outcome, it was reported that hypertensive disorders and birth of SGA fetus occurred in 62.5% and 73% women, respectively. In the present study, SGA babies were 21.6% with thick placenta diagnosed in the first trimester, hypertensive disorders occurred in 39.2%, and, of these, 22 (38%) had FGR also. Thame *et al.*,<sup>[26]</sup> in their study of placental volume in the second trimester and infant size at birth reported that placental volume is a very strong determinant of birth weight. Hafner *et al.*,<sup>[27]</sup> reported that placental growth between 12 and 22 weeks is too heterogeneous to justify its use as a clinical tool, but authors mention that it can provide new information on placental physiology, underlying unfavorable outcomes. Burton and Jauniaux<sup>[28]</sup> reported that there are two broad categories of preeclampsia, maternal, and placental. In placental preeclampsia, the problem arises from the placenta that is under hypoxia with oxidative stress. Placental preeclampsia appears to progress in two stages: Preclinical, clinical, and other studies of placenta from early gestation can help.

In the present prospective study of selected primigravida,



USG was done around 10 weeks and maternal neonatal outcome was studied. The findings suggest that the first trimester USG could help in early identification of risk, as placental location and dimensions seem to affect pregnancy outcome. Hence, it is recommended to have more studies regarding early gestation placental location and dimensions and there is a need for establishment of standards for estimation of placental volume and associated overall pregnancy outcome.

## STRENGTHS AND WEAKNESSES OF THE STUDY

Strengths of the study are inclusion of birth outcome in primigravida only, and the large numbers and follow-up until 1 week of birth.

Weaknesses of the study are lateral extent of placental location that are not included as well as details about possibility of placental migration. Also, only gross details included and intrinsic abnormalities of placenta were not included, but gross problems were automatically excluded.

## CONCLUSION

Placental location and dimensions in early gestation seem to indicate possibilities of future problems. Anterior placenta seems to be safe and fundal placenta is very dangerous. However, more studies are needed with details of lateral extension and follow-up of cases including hypertensive disorders, abruption, growth retardation, and cesarean section for fetal distress.

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## REFERENCES

1. Hoogland HJ, de Haan J, Martin CB Jr. Placental size during early pregnancy and fetal outcome: A preliminary report of a sequential ultrasonographic study. *Am J Obstet Gynecol* 1980;138:441-3.
2. Wolf H, Oosting H, Treffers PE. Second-trimester placental volume measurement by ultrasound: Prediction of fetal outcome. *Am J Obstet Gynecol* 1989;160:121-6.
3. Jauniaux E, Ramsay B, Campbell S. Ultrasonographic investigation of placental morphologic characteristics and size during the second trimester of pregnancy. *Am J Obstet Gynecol* 1994;170 (1 Pt 1):130-7.
4. Clapp JF 3<sup>rd</sup>, Rizk KH, Appleby-Wineberg SK, Crass JR. Second-trimester placental volumes predict birth weight at term. *J Soc Gynecol Invest* 1995;2:19-22.
5. Hayder M, Ali A. Ultrasonographic assessment of embryonic, fetal and placental development in Ossimi sheep. *Small Rum Res* 2003;73:277-82.
6. Ghoneim MR, Megahed H, Habba M, El-Biely MM, Lotfy GL. Diagnosis

- and prognostic value Doppler flow velocity waveform in high risk pregnancies. *Ultrasound Obstet Gynecol* 2008;18:40.
7. Tongsong T, Boonyanurak P. Placental thickness in the first half of pregnancy. *J Clin Ultrasound* 2004;32:231-4.
8. Gabbe SG, Niebyl JR, Simpson JL. *Obstetrics, normal and problem pregnancies*, 4<sup>th</sup> ed. New York: Churchill Livingstone; 2001.
9. Stables D, Rankin J. *Physiology in childbearing with anatomy and related biosciences*. Edinburgh: Bailliere Tindall; 2004.
10. Coall DA, Charles AK, Salafia CM. Gross placental structure in a low-risk population of singleton, term, first-born infants. *Pediatr Dev Pathol* 2009;12:200-10.
11. Kinare AS, Natekar AS, Chinchwadkar MC, Yajnik CS, Coyaji KJ, Fall CH, et al. Low midpregnancy placental volume in rural Indian women: A cause for low birth weight? *Am J Obstet Gynecol* 2000;182:443-8.
12. Thame M, Osmond C, Bennett F, Wilks R, Forrester T. Fetal growth is directly related to maternal anthropometry and placental volume. *Eur J Clin Nutr* 2004;58:894-900.
13. Gonser M, Tillack N, Pfeiffer KH, Mielke G. Placental location and incidence of pre-eclampsia. *Ultraschall Med* 1996;17:236-8.
14. Magann EF, Doherty DA, Turner K, Lanneau GS Jr, Morrison JC, Newnham JP. Second trimester placental location as a predictor of an adverse pregnancy outcome. *J Perinatol* 2007;27:9-14.
15. Cheng WW, Lin SQ. Analysis of risk factors for uteroplacental apoplexy complicating placental abruption. *Zhonghua Fu Chan Ke Za Zhi* 2008;43:593-6.
16. Kalanithi LE, Illuzzi JL, Nossov VB, Frisbaek Y, Abdel-Razeq S, Copel JA, et al. Intrauterine growth restriction and placental location. *J Ultrasound Med* 2007;26:1481-9.
17. Khan AT, Stewart KS. Ultrasound placental localization in early pregnancy. *Scott Med J* 1987;32:19-21.
18. Corneau J, Shaw L, Marchell CC, Lavery JP. Early placenta previa and delivery outcome. *Obstet Gynaecol* 1983;61:577-80.
19. Leiberman JR, Fraser D, Kasis A, Mazor M. Reduced frequency of hypertensive disorders in placenta previa. *Obstet Gynaecol* 1991;77:83-6.
20. Townsend RR, Laing FC, Nyberg DA, Jeffrey RB, Wing VW. Technical factors responsible for "placental migration": Sonographic assessment. *Radiology* 1986;160:105-8.
21. Redline RW. Placental pathology: A systemic approach with clinical correlations. *Placenta* 2008;29:S86-91.
22. Schwartz N, Wang E, Parry S. Two-Dimensional sonographic placental measurements in the prediction of small for gestational age infants. *Ultrasound Obstet Gynecol* 2012;40:674-9.
23. Elchalal U, Ezra Y, Levi Y, Bar-Oz B, Yanai N, Intrator O, et al. Sonographically thick placenta: A marker for increased perinatal risk--a prospective cross sectional study. *Placenta* 2000;21:268-72.
24. Willams MA, Hickokm DE, Zingheim RW, Luthy DA, Kimelman J, Nyberg DA, et al. Elevated maternal serum alpha-feto protein levels and mid trimester placental abnormalities in relation to subsequent adverse pregnancy outcomes. *Am J Obstet Gynaecol* 1992;167:1032-7.
25. Luigi R, Fabio G, Antonella C, Marthias N, Peter D. Prenatal diagnosis official journal of the internacional society for prenatal diagnosis 2004;24:182-8.
26. Thame M, Osmond C, Wilks R, Bennett FI, Forrester TE. Second-trimester placental volume and infant size at birth. *Obstet Gynaecol* 2001;98:279-83.
27. Hafner E, Metzzenbauer M, Höfingler D, Munkel M, Gassner R, Schuchter K, et al. Placental growth from the first to the second trimester of pregnancy in SGA fetuses and preeclamptic pregnancies compared to normal fetuses. *Placenta* 2003;24:336-42.
28. Burton GJ, Jauniaux E. Placental oxidative stress: From miscarriage to preeclampsia. *J Soc Gynaecol Invest* 2004;11:342-52.

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